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Epitope based vaccine prediction for SARS-COV-2 by deploying immuno-informatics approach

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ABSTRACT

A new virus termed SARS-COV-2 (causing COVID-19 disease) can exhibit a progressive, fatal impact on individuals. The World Health Organization (WHO) has declared the spread of the virus to be a global pandemic. Currently, there are over 1 million cases and over 100,000 confirmed deaths due to the virus. Hence, prophylactic and therapeutic strategies are promptly needed. In this study we report an epitope, ITLCFTLKR, which is biochemically fit to HLA allelic proteins. We propose that this could be used as a potential vaccine candidate against SARS-COV-2. A selected putative epitope and HLA-allelic complexes show not only better binding scores, but also RMSD values in the range of 0–1 Å. This epitope was found to have a 99.8% structural favorability as per Ramachandran-plot analysis. Similarly, a suitable range of IC_{50} values and population coverage was obtained to represent greater validation of T-cell epitope analysis. Stability analysis using MDWeb and half-life analysis using the ProtParam tool has confirmed that this epitope is well-selected. This new methodology of epitope-based vaccine prediction is fundamental and fast in application, ad can be economically beneficial and viable.

1. Introduction

The outbreak of COVID-19 in the Hubei region of the Chinese city of Wuhan [37] has resulted in a difficult situation for the global populace and for the World Health Organization (WHO). On 30 Jan 2020, WHO declared an emergency regarding COVID-19 spread, prevention, and control [28,29]. As of 20 March 2020, there are over 240,000 cases and over 10,000 confirmed deaths, affecting 181 countries [9]. Present updates indicate that there are over 1 million confirmed cases and over 100,000 deaths worldwide [39] (information is obtained from https://www.worldometers.info/coronavirus/). The coronavirus is an RNA type virus with positive sense strand feature and is associated with the Coronaviridae family under order Nidovirales, and is found to be dispersed among Primate order, members of class Mammalia, and specifically in humans [31]. Severe acute respiratory syndrome coronavirus (SARS-CoV) [11,21,22] and Middle East respiratory disorder coronavirus (MERS-CoV) [7,40], classified as β-coronaviruses, are found to be related to the novel SARS-COV-2. Until now, there has been no efficacious therapy to regulate its spread [24]. Investigating its spread across continents and the vulnerability, there is an urgent need to craft vaccines for reinforcing immune defense against the SARS-COV-2 virus. One of the strategies to combat it is the development of vaccines that can initiate an adaptive immune response in humans. Here an attempt has been made to design an epitope based vaccine directed at SARS-COV-2, by analyzing the proteome of the virus by using *Immuno-informatics* tools. In this study, we deployed the use of various bioinformatics servers and Immuno-informatics tools for identifying and recognizing the T-cell epitopes from the intensive study of available protein sequences and structures that are related to SARS-COV-2. These epitope stretches can interact with MHC Class I and Class II HLA alleles; further validation of epitopes was analyzed by Ramachandran Plot analysis, Antigenicity parameters evaluation, Toxicity analysis, Population coverage, Molecular dynamics, and ProtParam analysis [19]. This approach is an excellent method in modern vaccine design, as it provides a lead over classical trial and error methods of wet labs [23]. We tried to identify T-cell epitopes that can elicit a robust immune response in the global human population and act as potential vaccine candidates. However, the ability of these epitopes to act as a vaccine candidate needs to be analyzed in Molecular biology lab studies. Our investigation can open new dimensions in crafting peptide-based vaccine regimens for Novel SARS-COV2. The greatest decline in virus expansion was noted following ORF3a removal. ORF7a encodes a 122-amino-acid type I

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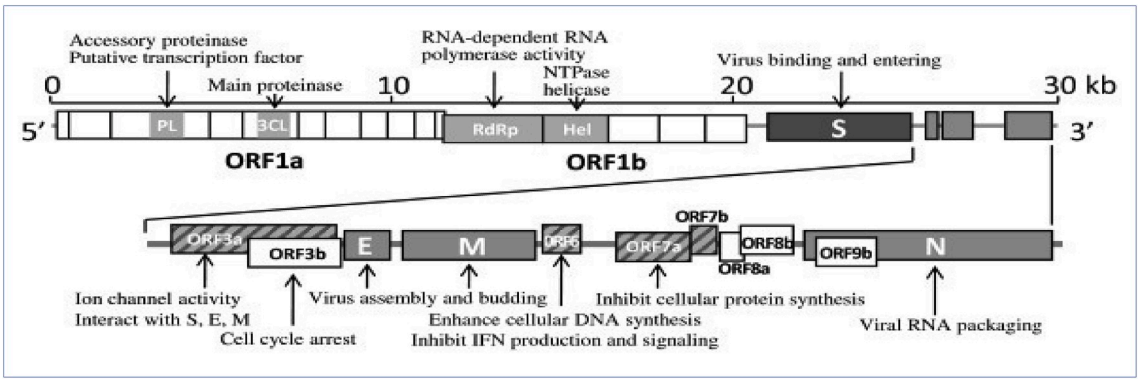


Fig. 1. Genome organization and viral proteins of SARS-CoV [44].

Table 1
List of proteins selected for SARS COV-2 with allergenicity.

Protein Gene Bank accession no.	Protein name	Allergen FP Score (Tanimoto similarity Index)	Allergen/ Non-Allergen
QHD43418.1	Envelop Protein	0.87	Non-Allergen
QHD43417.1	ORF3a Protein	0.84	Non-Allergen
QHD43423.2	Nulceocapsid Phosphoprotein	0.85	Non-Allergen
QHD43421.1	ORF7a protein	0.80	Non-Allergen
QHD43419.1	Membrane Glycoprotein	0.83	Non-Allergen

transmembrane protein and structural studies disclose a packed seven-stranded β sandwich comparable in fold and topology to members of the immunoglobulin super family. SARS-CoV is an enveloped, positive-stranded RNA virus with a genome of around 29,700 bases. The genome incorporates at least 14 open reading frames (ORFs) that encode 28 proteins in three distinct classes: two large polyproteins P1a and P1ab that are cleaved into 16 non-structural proteins (nsp1–nsp16) during viral RNA synthesis; four structural proteins (S, E, M and N) that are necessary for viral entrance and gathering; and eight accessory proteins that are assumed to be dispensable for viral replication, but may facilitate viral assembly and take part in virulence and pathogenesis (Fig. 1).

In our investigatory study, out of five, two proteins namely ORF-3a and ORF-7a specific to SARS-COV-2 were found to be putative T-cell epitope determinants that create useful information to distinguish, and these proteins are also important for viral replication and growth [41]. Both of these proteins may influence in viral pathogenesis and disease spread, although the literature lacks unanimity [43]. B-cell epitopes prediction is still considered to be untrustworthy for both linear and conformational epitopes as compared to T-cell epitopes. Furthermore, the B-cell epitopes do not elicit a strong antibody response. For this reason, only T-cell epitopes are considered in the present study. It is capable to produce $CD4^+$ and $CD8^+$ T-cells with long-lasting response [45]. . In one of the recent studies, epitopes were designed, but they have focused on the single protein (i.e. SPIKE protein) to generate multiple epitopes like 13 for MHC I and 3 for MHC II [46]. We have analyzed multiple proteins to screen only effective epitopes based on various *in-silico* filters, to provide the most appropriate and authentic epitopes, which can be further tested in a wet lab.

2. Methodology

2.1. Protein retrieval and allergenicity analysis

Five protein sequences were selected from NCBI-GenBank, listed in Table 1, for SARS-COV-2 based on their Allergenicity that relies on the Tanimoto similarity index score produced by AllergenFP 1.0 [8]. The selected proteins were the envelope protein, ORF3a protein, nucleocapsid phosphoprotein, ORF7a protein, and membrane glycoprotein, which are crucial for the structural integrity and functionality of the virus [38].

2.2. T-cell epitope prediction for MHC HLA alleles

IEDB (Immune epitope database) [20] along with NetMHCII PAN 3.2 and NETMHC 4.0 servers [18] were effectively used for finding putative peptide sequences that were aimed to interact with the MHC Class II and I HLA alleles, respectively (because of efficient algorithms based on artificial neural networks). The VaxiJen score is determined for screening the best antigenic epitopes using the VaxiJen online tool [10] with a threshold $\geq$ 1.0 for the viruses' domain.

2.3. Structural prediction: Putative epitopes and MHC HLA alleles

The epitopes 3D structural findings were conducted by using the PEP-FOLD-3.5 server [25,33,34] and MHC HLA Allelic peptides tertiary or 3D structure were obtained from the RCSB-PDB database [4].

2.4. Molecular docking analysis

The selected epitopes and HLA complexes were docked for calculating refined interactions and binding energies, along with atomic contact energy (ACE), by using two docking web-servers: DINC 2.0 [2] and PatchDock [32].

2.5. Molecular dynamics-simulation analysis of docked complex

Molecular dynamics study was conducted to analyze RMSD values and atomic fluctuations for all amino acids under the 100 ps time frame by deploying the MDWeb server. MDWeb server was deployed to analyze Coarse grained MD Brownian dynamics (C-alpha) with specifications → Time: 100 ps, output frequency (steps) = 10, force constant (kcal/mol Á^2) = 40, distance between alpha carbon atoms(Á) = 3.8 for both the interacting epitopes, and it was based on a GROMACS MD setup with solvation using an Amber-99sb* force-field [17].

2.6. Toxicity, Ramachandran-plot, and population coverage analysis

The ToxinPred server [16] is utilized for determining the toxicity

Table 2
Probable antigenic epitopes and MHC I Allele interaction based on NETMHC 4.0 server and VaxiJen 2.0 scores (≥1.0) for antigenicity prediction. (* - sign is used for no interaction any HLA Allele under consideration).

NCBI-GenBank ID	MHC I Allele	POS	Core peptide	1-LOG50K	Affinity(nM)	Vaxijen score	Antigenicity
QHD43417.1	HLA-A*68:01	7	FTIGTVTLK	0.853	4.9	2.0317	ANTIGEN
	HLA-A*31:01	125	RLWLCWKCR	0.74	16.59	1.1604	ANTIGEN
QHD43418.1	–	–	–	–	–	–	NO INTERACTION
QHD43419.1	HLA-A*11:01	5	GTITVEELK	0.719	20.98	1.0976	ANTIGEN
QHD43421.1	HLA-A*11:01	109	ITLCFTLKR	0.71	22.97	2.0208	ANTIGEN
	HLA-A*68:01	109	ITLCFTLKR	0.695	27.24	2.0208	ANTIGEN
	HLA-A*23:01	107	VFITLCFTL	0.621	60.42	1.2490	ANTIGEN
QHD43423.2	–	–	–	–	–	–	NO INTERACTION

Table 3
Probable antigenic epitopes and MHC II Allele interaction based on NETMHC II PRED 3.2 server and VaxiJen 2.0 scores (≥1.0) for antigenicity prediction. (* - sign is used for no interaction any HLA Allele under consideration).

NCBI-GenBank ID	MHC II Allele	POS	Core peptide	1- LOG50K	Affinity(nM)	Vaxijen score	Antigenicity
QHD43417.1	–	–	–	–	–	–	NO INTERACTION
QHD43418.1	–	–	–	–	–	–	NO INTERACTION
QHD43419.1	HLA-DRB1*04:01	55	WLLWPVTA	0.282	2375.78	1.0631	ANTIGEN
QHD43421.1	HLA-DRB1*01:01	74	VYQLRARSV	0.484	267.05	1.3108	ANTIGEN
	HLA-DRB1*07:01	74	VYQLRARSV	0.342	1235.08	1.3108	ANTIGEN
QHD43423.2	–	–	–	–	–	–	NO INTERACTION

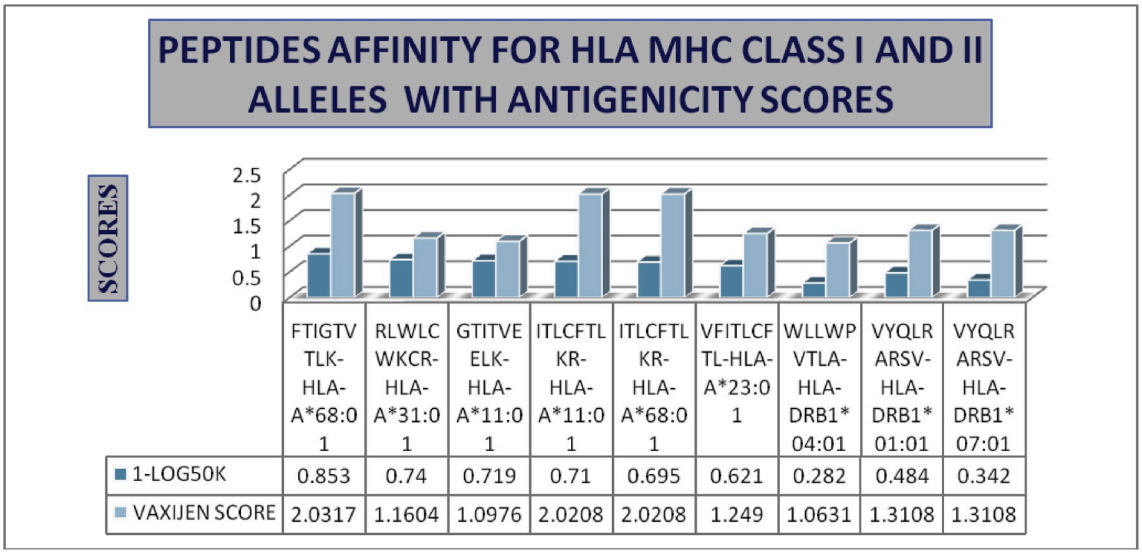


Fig. 2. Graphical representation of selected peptides for docking based on their interaction with MHC Class I and Class II HLA alleles along with their Antigenicity.

scoring of Epitopes for selecting non-toxic ones; also, the Ramachandran plot analysis was deployed by using the MolProbity 4.2 server [6] to analyze the quantitative presence of residues in the favorable region.

The Immune Epitope Database (IED) resource web-server of population coverage was used to predict population coverage of the MHC II and MHC I alleles that interact with screened out epitopes based on their restriction database [5]. The MHCPred web-server was effectively used in quantitative prediction of sorted out epitopes interacting with HLA alleles of MHC II and MHC I [15]. Thereafter the ProtParam tool [42] of the ExPASy server was used to screen final stable epitopes based on the instability index and half-life.

Table 4
Listing of MHC HLA-Alleles respective Crystal structures/Models with the PDB ID.

Allele Name	Template structure (PDB-ID)	Crystal structure/Model
HLA-A*11:01	2HN7	CRYSTAL STRUCTURE
HLA-A*23:01	3I6L	CRYSTAL STRUCTURE
HLA-A*31:01	3RL1	CRYSTAL STRUCTURE
HLA-A*68:01	6PBH	CRYSTAL STRUCTURE
HLA-DRB1*01:01	4AH2	CRYSTAL STRUCTURE
HLA-DRB1*04:01	5LAX	CRYSTAL STRUCTURE
HLA-DRB1*07:01	6BIJ	CRYSTAL STRUCTURE

3. Results

3.1. T-cell epitopes prediction and VaxiJen scoring

Non-allergen proteins selected (Table 1) based on allergenicity scores (by deploying AllergenFP 1.0) New to NetMHCII PAN 3.2 and NETMHC 4.0 servers for determining 1-log50k value and affinity values for selecting the best possible pair of epitopes with their corresponding HLA alleles. In Table 2 and Table 3, results for MHC Class I HLA and MHC Class II HLA alleles paired epitopes along with their VaxiJen scores were represented respectively, to obtain putative epitopes. The results are self-explanatory in Fig. 2. Graphical representation of selected

Table 5
Binding Energies, Ace Values for Docked Complexes based on DINC Server and PatchDock Analysis for Putative Epitopes.

Epitope	HLA Allele	Binding score (kcal/mol)	Patch dock score	ACE	Selection
FTIGTVTLK	HLA-A*68:01	−8.80	8066	178.91	Selected
RLWLCWKCR	HLA-A*31:01	−4.80	8916	117.53	Rejected
GTITVEELK	HLA-A*11:01	−3.80	8040	78.78	Rejected
ITLCFTLKR	HLA-A*11:01	−3.70	8206	−25.88	Selected
ITLCFTLKR	HLA-A*68:01	−7.60	8136	184.55	Selected
VFITLCFTL	HLA-A*23:01	−4.40	7706	−134.91	Rejected
WLLWPVTLA	HLADRB1*04:01	−8.60	9432	−150.96	Rejected
VYQLRARSV	HLADRB1*01:01	−6.20	6874	−168.74	Selected
VYQLRARSV	HLADRB1*07:01	−6.20	6842	262.74	Selected

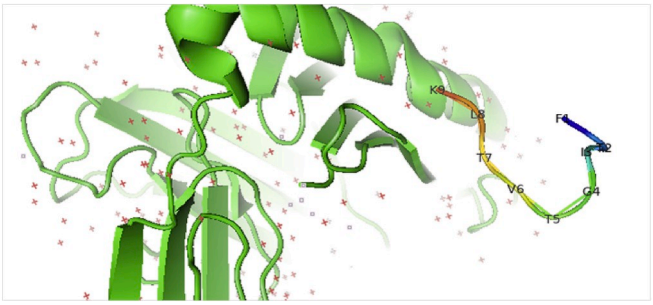


Fig. 3. FTIGTVTLK Epitope interaction with an antigen-binding pocket of HLA-A*68:01, of MHC I-HLA Allele. Here, Threonine at 2nd,5th, and 7th position in the epitope generate preferably a hydrogen bond due to the presence of partially charged positive and negative atoms, and also 4th position Glutamic acid and lysine at 8th position side chains can form a salt bridge, while other amino acids result in van der Waals interactions.

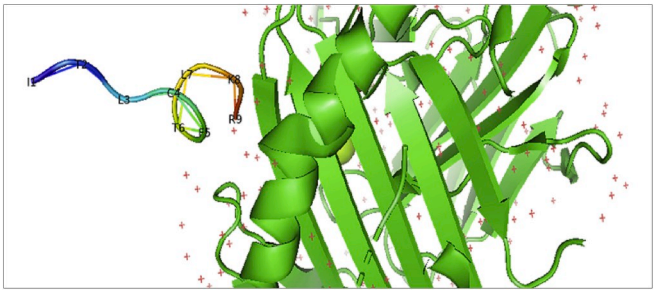


Fig. 4. ITLCFTLKR Epitope interaction with an antigen-binding pocket of HLA-A*68:01, of MHC I-HLA Allele. Here, Threonine at 2nd and 6th position, as well as cysteine at the 4th position in epitope, generate preferably a hydrogen bond due to the presence of partially charged positive and negative atoms, while other amino acids result in van der Waals interactions.

peptides for docking are based on their interaction with MHC Class I and Class II HLA alleles along with their antigenicity.

3.2. Structural findings of epitope and MHC HLA-Alleles

Epitope structures were obtained by using the PEP-FOLD-3.5 webserver; the HLA allele's structures were retrieved from the RCSB-PDB database. In Table 4 the crystal structure/model structure details with reference PDB-Id is provided.

3.3. Molecular docking analysis

It was found that FTIGTVTLK, ITLCFTLKR epitopes interacted with

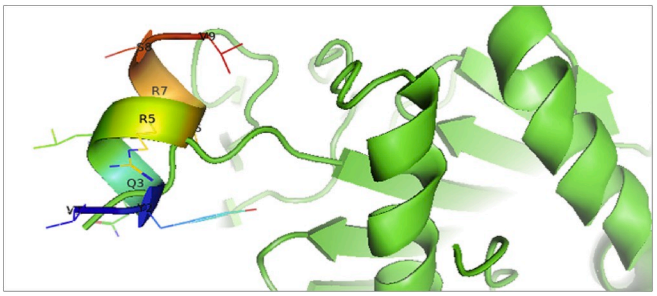


Fig. 5. VYQLRARSV Epitope interaction with an antigen-binding pocket of HLA-DRB1*07:01, of MHCII-HLA Allele, Here, Tyrosine at 2nd, Glutamine at 3rd position and Serine at 8th position in epitope generate preferably a hydrogen bond due to the presence of partially charged positive and negative atoms, and 5th and 7tharginine residue side chains can form a salt bridge, while other amino acids result in van der Waals interactions.

MHC class I HLA Alleles, and VYQLRARSV epitope interacted with MHC Class II HLA alleles with a perfect binding score and ACE values as shown in Table 5. The ITLCFTLKR Epitope of the ORF-7A protein exhibits binding with 2 HLA alleles (HLA-A*11:01, HLA-A*68:01) of MHC Class I, while FTIGTVTLK Epitope of ORF-3a protein interact with 1 HLA Allele (HLA-A*68:01) of MHC Class I. The VYQLRARSV Epitope of ORF-7a protein interacts clearly with 2 HLA Alleles (HLA-DRB1*01:01, HLA-DRB1*07:01) of the MHC Class II domain.

In Figs. 3, 4, and 5, interactions between a selected three T-Cell epitopes with respective MHC Class I and II HLA-Alleles via hydrogen bond formation and van der Waals interactions is depicted. After positive docking results, these epitopes were subjected to further Molecular dynamic simulation and biochemical parameters assessment. Fig. 6 represents a graphical plot of binding scores for epitopes interacting with HLA-Alleles.

3.4. Molecular dynamics and simulation analysis

RMSD values and Atomic fluctuation per amino acid residue were obtained for Epitopes interacting with the HLA-Allele structure; this analysis allows a perfect pair selection and validation. Moreover, only two Epitope pairs, i.e., ITLCFTLKR and VYQLRARSV, were identified as probable T-cell epitopes and as putative vaccine specimens. Fig. 7 shows the RMSD Plot and Atomic fluctuation per residue for the ITLCFTLKR-HLA-A*68:01 complex, the RMSD Plot and Atomic fluctuation per residue for the VYQLRARSV- HLA-DRB1*07:01 complex. Both results were positive as best interactions, for protein-ligand docked complexes must possess RMSD values from 0 to 1.0 Å as a preferred range [13].

3.5. Toxicity analysis, Ramachandran Plot analysis, and population coverage results

ToxinPred 4.0 server results (in Table 6.) represent Finalized T-cell Epitopes that were nontoxic from the biochemical perspective.

Ramachandran plot analysis, Fig. 8A and B suggest that most of the residues are allowed in a favored region; this gives more confidence in the structural conformation for targeted T-Cell Epitopes.

MHCPred results (Table 7) indicate quantitative estimation of IC_{50} values for both MHC I and MHC II alleles for respective Epitopes shows elicitation of an immune response when this data is deployed in a population coverage analysis.

IEDB population coverage analysis suggests that ITLCFTLKR and VYQLRARSV epitopes exhibit a suitable population coverage, as depicted in the graphical representation of Fig. 9A and B. This allows only two probable Epitopes for the final selection of vaccine crafting.

In Table 8, ProtParam analysis further reveals the stability of the considered epitopes and final revelation of one epitope ITLCFTLKR is screened out. This particular Epitope exhibits an instability index of

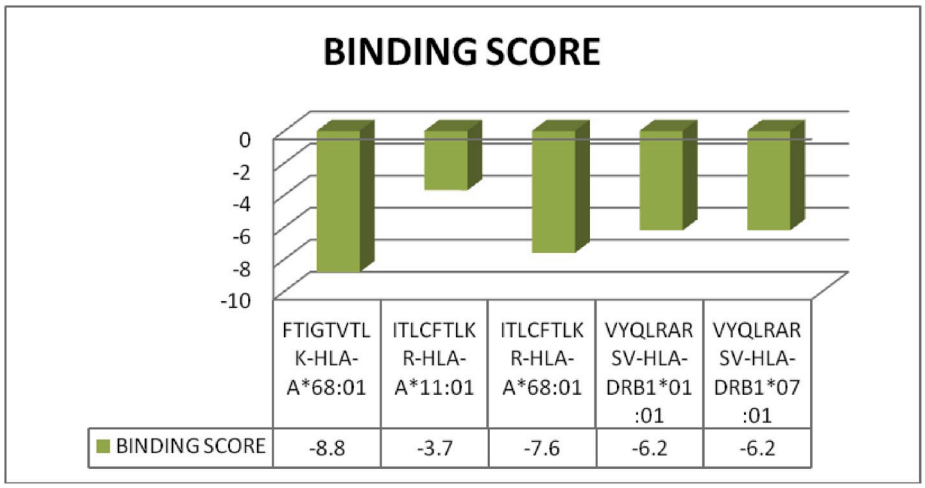


Fig. 6. Binding energy graphical plot for selected Epitope and HLA-Allelic pair.

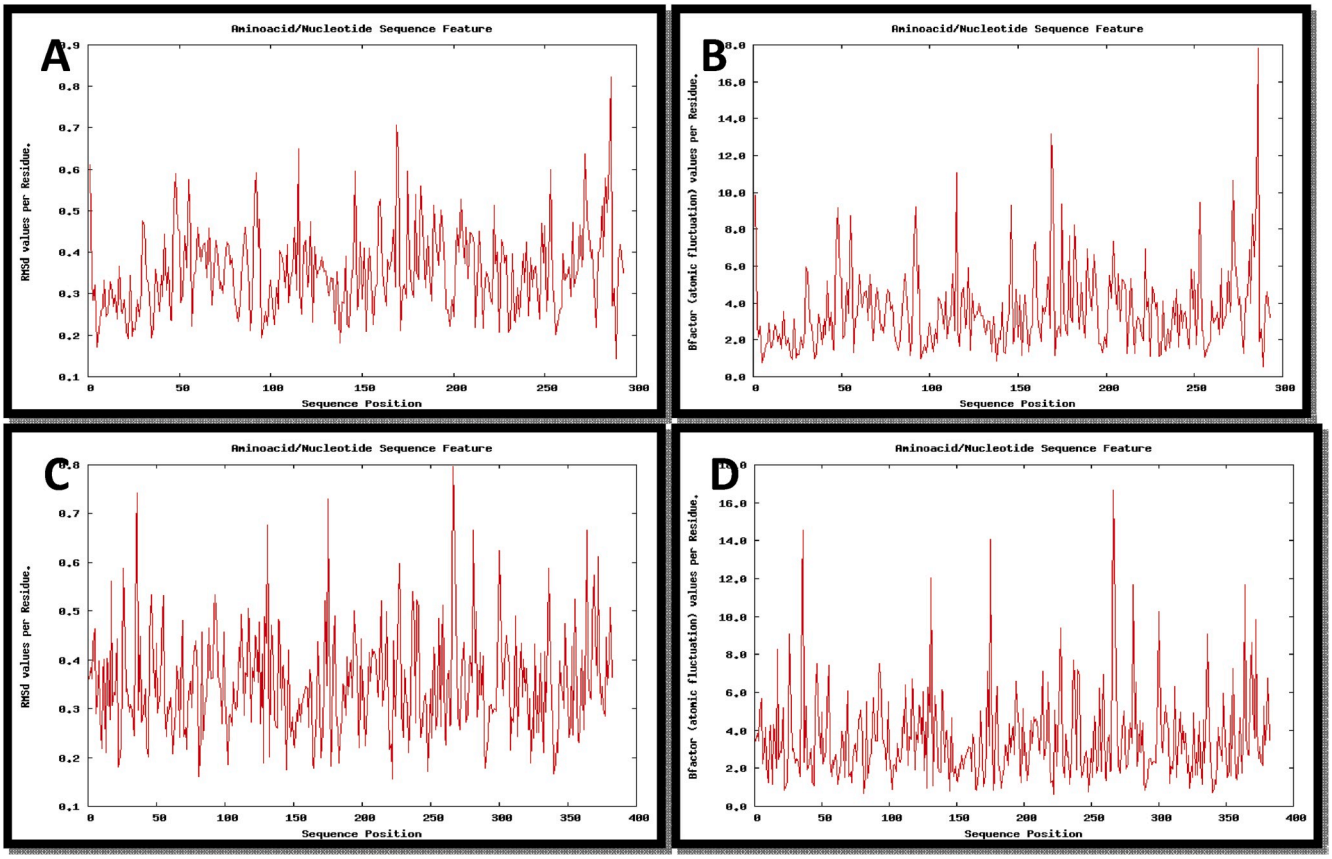


Fig. 7. A. RMSD Plot for ITLCFTLKR- HLA-A*68:01 complex, for each amino acid residue by Molecular dynamics analysis, B. B-Factor (atomic fluctuation) values per amino acid residue for Epitope ITLCFTLKR- HLA-A*68:01 docked complex, C. RMSD Plot for VYQLRARSV- HLA-DRB1*07:01, for each amino acid residue by Molecular dynamics analysis, D. B-Factor (atomic fluctuation) values per amino acid residue for Epitope VYQLRARSV- HLA-DRB1*07:01 docked complex.

35.68, with a grand average of hydropathicity (GRAVY) calculated was 0.844, and the estimated half-life for this peptide was determined to be 20 h for mammalian reticulocytes.

4. Discussion

In this study, SARS-COV-2 virus proteins were analyzed by using *In-silico* methods, and can be further utilized for vaccine trials as per earlier successes in the case of similar SARS-COV studies, and later observed in the development of polyclonal antibodies [27]. Here we obtained two epitopes ITLCFTLKR and VYQLRARSV after successful docking and molecular dynamics simulation; furthermore, these two epitopes were subjected to population coverage and toxicity analysis. Similarly, in another study, for MERS-COV, nucleocapsid peptides were used for T-cell epitope prediction, and found to be successful [36]. The IEDB and NCBI-GenBank database were fully deployed to analyze sequence homology, to predict targets for COV-2 in case of viral protein identification as per the related studies [14], as ViPR (Virus Pathogen database

Table 6
Results of ToxinPred on probable antigens.

Peptide/Probable antigen	SVM score	Hydrophilicity	Molecular weight	Toxicity
FTIGTVTLK	−1.36	−1.23	979.32	NON-TOXIN
GTITVEELK	−0.98	0.34	989.27	NON-TOXIN
ITLCFTLKR	−1.32	−0.41	1094.51	NON-TOXIN
VFITLCFTL	−1.21	−1.52	1056.46	NON-TOXIN
WLLWPVTLA	−1.18	−1.62	1098.49	NON-TOXIN
VYQLRARSV	−1.07	−0.12	1091.40	NON-TOXIN

analysis resource) are also dependent on IEDB and GenBank primarily [30]. We analyzed five different proteins in SARS-COV-2 for the present study (because of their availability in the NCBI-GenBank database and importance in a structural role in SARS-COV-2 [14] and finally revealed T–Cell epitopes that can be used for wet lab considerations and time savings. In a very recent study, different epitopes were found for SARS-COV-2, based on In-silico approaches and focused on only surface glycoprotein [3], but in our research study there are many differences as we analyzed a different group of proteins from SARS-COV-2 to sort out short length T-Cell epitopes specific to MHC I as well as MHC II diversified HLA-Alleles.

It is reported for SARS-CoV HLA-B*4601, HLA-B*0703, HLA-DR B1*1202 are activated [26], interaction with different MHC I and II allelic forms namely HLA-A*11:01, HLA-A*68:01, HLA-DRB1*01:01 and HLA-DRB1*07:01. $CD4^{+}$ and $CD8^{+}$ memory T cells. Based on prior literature, it is anticipated that it can persist for four years as in the case of SARS-CoV recovered individuals, show T-cell proliferation, DTH response, and production of IFN-γ [12]. We surmise that our screen can be more effective and useful. Primarily molecular docking reveals three Epitopes, but as we proceed to Molecular dynamic simulations, it reveals best interactions for two epitopes i.e., ITLCFTLKR and VYQLRARSV, with acceptable stability analyzed with the help of MDWeb and identified by using best available tools with easy-to-apply methods. One recent study was found to be focused on developing monoclonal

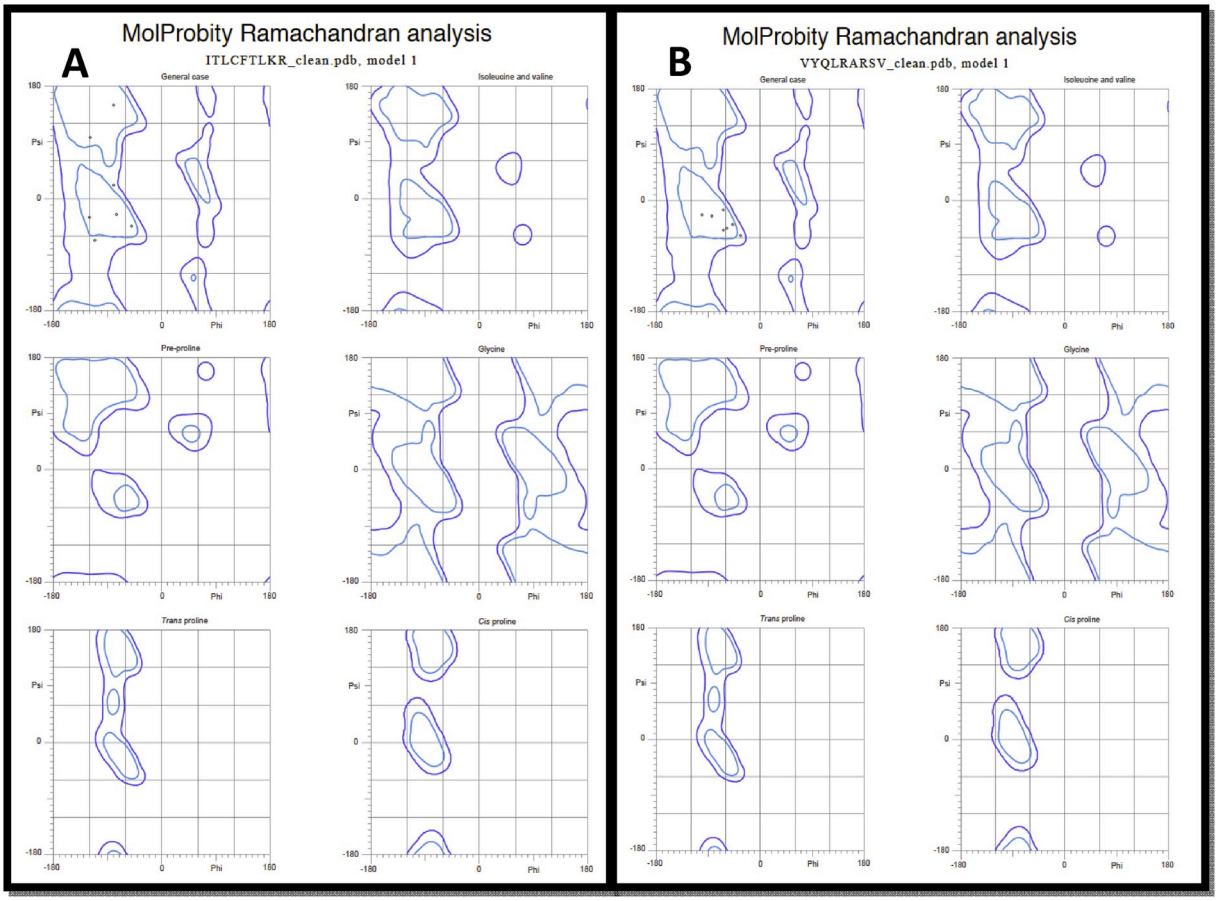
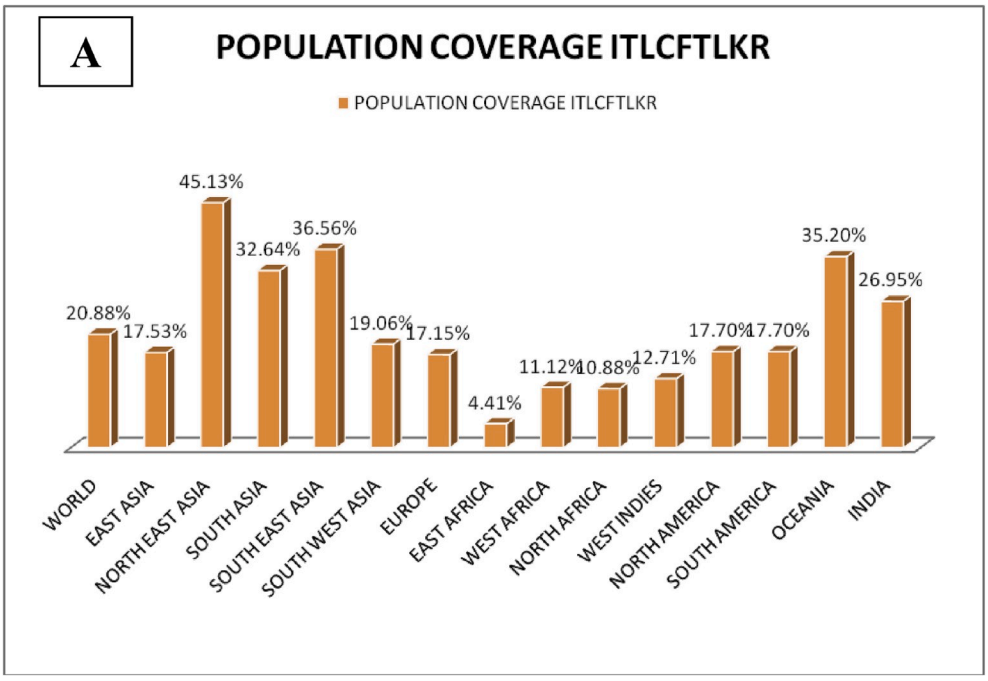


Fig. 8. A. 99.8% residues of the ITLCFTLKR Epitope were in the allowed and favored region under Ramachandran Plot analysis. B. 99.8% residues of the VYQLRARSV Epitope were in the allowed and favored region under Ramachandran Plot analysis.

Table 7
MHCPred results depict IC_{50} Values for HLA Alleles and confidence of the prediction.

HLA Alleles	Amino acid groups	Predicted -logIC_{50} (M)	Predicted IC_{50} Value (nM)	Confidence of prediction (Max = 1)
HLA-A*68:01	FTIGTVTLK	7.116	76.56	1.00
HLA-A*11:01	ITLCFTLKR	7.028	93.76	1.00
HLA-A*68:01	ITLCFTLKR	6.282	522.40	0.78
HLA-DRB1*01:01	VYQLRARSV	7.624	23.77	0.89
HLA-DRB1*07:01	VYQLRARSV	6.734	184.50	0.89



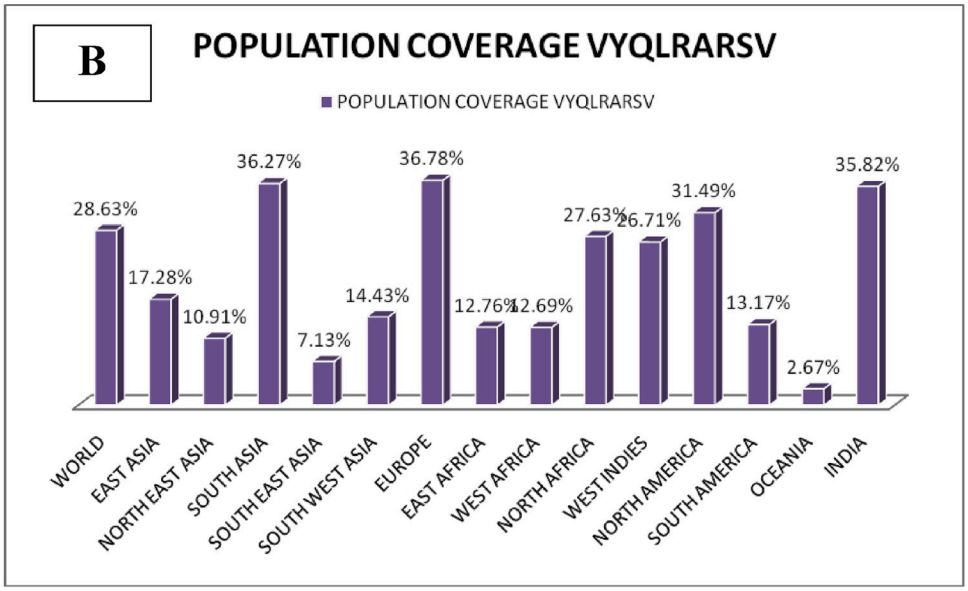


Fig. 9. A. Graphical representation of population conservancy analysis of ITLCFTLKR Epitope. B. Graphical representation of population conservancy analysis of VYQLRARSV Epitope.

Table 8
ProtParam analysis for selected epitopes.

Selected Epitope	GRAVY Score	Instability Index (Indication)	Estimated Half-Life(Mammalian reticulocytes)	Theoretical pI	Aliphatic Index
ITLCFTLKR	0.844	35.68(Stable)	20 Hours	9.51	130.00
VYQLRARSV	−0.067	70.73(Unstable)	100 Hours	10.83	118.89

antibodies like CR-3022 against the Spike protein of SARS-COV-2 that also exhibits interaction with ACE (Angiotensin Converting Enzyme) enzyme of the Human respiratory epithelium and requires complex neutralizing mechanisms for several binding domains [35], whereas in our study the putative T-cell epitopes can directly interact with MHC-Allelic sets that can be useful for developing immunization against SARS-COV-2. ProtParam [42] analysis further reveals the stability of the considered epitopes, and final revelation of one epitope ITLCFTLKR is screened out. This particular Epitope shows an instability index of 35.68 with a grand average of hydropathicity (GRAVY) calculated as 0.844, and the estimated half-life for this peptide was determined to be 20 h for mammalian reticulocytes.

Satisfactory population coverage was observed for targeted epitopes - HLA allelic complexes at the worldwide, South Asia, and India level. The biochemical integrity in epitope structure was further evident by deploying Ramachandran plot analysis. Both epitopes were non-toxic,

non-allergenic, and possess good antigenicity. In a similar study of the preliminary analysis of COVID-19 vaccine targets [1] the investigators tried to use the spike protein and nucleo-capsid protein sequences of SARS-COV that are homologous to some extant with SARS-COV-2 proteins to determine multiple different epitopes for Vaccine prediction, but in our study out of five two proteins namely ORF-3a and ORF-7a specific to SARS-COV-2 were found to be putative T-cell epitope determinants that create useful information; these proteins are also important for viral replication [41]. Both biochemical parameters, as well as an advanced HMM and ANN based algorithm in selected Immuno-informatics tools, were very useful to present a clear picture of predicted epitopes for crafting vaccine against SARS-COV-2. The only limitation that can be considered as future scope is that these easily synthesized peptides should be tested with *In-vitro* study for more practical validation.

5. Conclusion

ITLCFTLKR epitope was selected for crafting and designing a vaccine against SARS-COV-2. This particular epitope has good antigenicity, exhibits active binding with MHC HLA-Alleles, and has maximum population coverage for different geographical regions. Therefore, this peptide can be further used in vaccine design against SARS-COV-2 after wet lab verification. This novel approach can also assist life science research groups to reduce time, monetary expenditures, as well as physical hit-trial efforts.

Ethical approval

I confirm that authors did not perform any experiments on human or animals.

Declaration of competing interest

I confirm that the authors hereby declare they that have no conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.imu.2020.100338.

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